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ROLE OF BODY FAT IN THE PREDICTION OF THE METABOLIC  
RESPONSE FOR IMMERSION IN COLD WATER

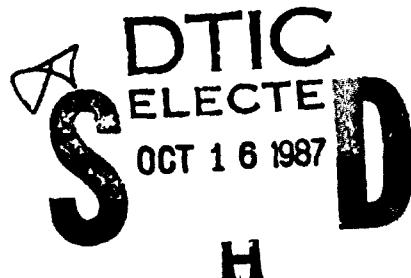
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Running Head: Role of body fat



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## ABSTRACT

Several empirical models for predicting the metabolic response to a lowered body temperature have been evaluated against available data of young healthy males immersed in cold water under resting conditions. Nude immersions took place in 20 and 24°C water for 1 h and clothed immersions took place in 10 and 15°C for 3 h. The data were pooled according to low and high percent body fat (%BF). Decreases in the mean weighted skin temperature ( $T_{sk}$ ) ranged from 5.3 to 11.9°C and decreases in the core temperature ( $T_c$ ) ranged from 0.56 to 1.54°C, while increases in the metabolic rate over the immersion period ranged from 34 to 256 W. Through regression analysis, an inverse relationship between %BF and the metabolic response for a given lowered  $T_{sk}$  and lowered  $T_c$  was established. When this relationship was explicitly applied to the models, significant improvements in their predictive capability were found. Variables such as body weight, body surface area and the rate of change of  $T_{sk}$  were not found to contribute to the predictive capability of the models. *Keywords:* modeling; response (biology); physiological effects; hypothermia ←

**Index Terms:** prediction model, cold water, immersion, shivering, body fat

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## INTRODUCTION

Several studies (1-5) have demonstrated that individual of low subcutaneous body fat (BF) cools more quickly than one of high BF when immersed in cold water. This increased rate of cooling is associated with an increased metabolic response in the form of shivering. Shivering is the body's method of generating heat to reduce or halt the rate of cooling. It is natural, therefore, to assume that an increase in metabolic rate correlates to a decrease in deep body temperature. Indeed, this has been the basis of several models that predict the metabolic response for exposure to cold.

Thermoregulatory signals that determine the metabolic response are thought to be generated by thermoreceptors located in the skin and the hypothalamus. In practice, a mean weighted skin temperature ( $T_{sk}$ ) is used to define the signal from the skin. To approximate the signal from the hypothalamus, the temperature of a particular core site such as the esophagus or rectum is used. In the present study, the temperature of these core sites will be represented by  $T_c$ .

The models that predict metabolic response from measurements of  $T_{sk}$  and  $T_c$  range from a simple linear dependence upon these two variables (6,7) to a more complex dependence based upon the differences between measured and fixed or set-point temperatures (8-11). Most models, however, do not differentiate the response for individuals of varying BF. That is, the predicted metabolic response is often a function of only  $T_{sk}$  and  $T_c$ , yet, for a given lowered  $T_{sk}$  and lowered  $T_c$ , there is evidence that an individual of low BF has a higher response than an individual of high BF. This stems from the early cold water immersion studies of Gee and Goldman (12) and Bynum and Goldman (13) which were later analyzed by Strong et al. (7) and from the data of McArdle et al. (14) and Toner et al. (15) used in the present study. Strong et al. (7) recognized the different metabolic responses

between individuals of varying morphology and proposed separate model equations for small-lean, average and heavy-fat individuals.

The present study does not address how the various thermoregulatory signals are generated or integrated to produce the observed metabolic responses; nor does it address whether, and if so how, these properties vary between individuals of different morphology. Instead, this study examines how well the above referenced models predict the metabolic response for a given lowered  $T_{sk}$  and lowered  $T_c$ . Using the more recent data of McArdle et al. (14) and Toner et al. (15), the predictive capability of these models are compared through regression analysis. It is then demonstrated that the predictive capability of these models can be improved by explicitly accounting for BF through an inverse relationship between the metabolic response and BF. Finally, a residual analysis of the best model predictions is presented to examine whether the assumption of normality has not been violated and whether the dependencies on  $T_{sk}$  and  $T_c$  have been accounted for adequately.

## METHODS

Data. Data were obtained from both nude ( $n=8$ ) and clothed ( $n=10$ ) young healthy males immersed in cold water under resting conditions. Nude (subjects wore a nylon swim suit) immersions took place in 20 and 24°C water for 1 h (14) and clothed immersions took place in 10 and 15°C water for 3 h (15). Clothing consisted of a neoprene dry suit (3 mm thickness) and one of two polypropylene undergarments with an average total insulative value (based on calibration with an immersed manikin) of 0.95 (0.15) and 1.28 clo (0.20  $\text{m}^2 \text{ }^\circ\text{C}/\text{W}$ ), respectively. Rectal and skin temperatures were measured with a rectal probe and waterproofed thermal sensors. Metabolic rates (MR) were determined by standard techniques of open-circuit spirometry.

All data were separated according to low (<14%) and high BF (individual %BF ranged from 3 to 23.1%) and grouped according to exposure. The number of subjects per group varied between 4 and 5. LBF and HBF are used to designate the low and high BF groups, respectively. Although each clothed subject was exposed to the two immersion temperatures of 10 and 15°C, not all completed the targeted duration of 3 h and, in some cases, data were incomplete (---). In these circumstances, the number of subjects in the group was reduced from 5 to 4. Group averages were adjusted accordingly when these data were applied to the models. For the LBF, these average values ranged as: %BF from 9.3 to 10.8, body weight (WT) from 69.8 to 72.4 kg, body surface area ( $A_D$ ) from 1.86 to 1.93 m<sup>2</sup>, decreases in  $T_{sk}$  from 5.3 to 11.9°C, decreases in  $T_c$  from 0.98 to 1.54°C, and mean increases in the metabolic rate above the pre-immersion resting value ( $\Delta MR$ ) from 52 to 256 W. For the HBF, average values ranged as: %BF from 16.8 to 19.7, WT from 74.3 to 80.0 kg,  $A_D$  from 1.85 to 1.97 m<sup>2</sup>, decreases in  $T_{sk}$  from 6.4 to 11.4°C, decreases in  $T_c$  from 0.56 to 1.09°C, and  $\Delta MR$  from 34 to 86 W. Rates of change with time of  $T_{sk}$  and  $T_c$  were approximated by linear interpolation.

Models. The models examined are listed in Table 1 and although this list is not exhaustive, it does represent a wide cross-section of available models. The first three models predict MR and use the rectal temperature to represent  $T_c$  while the remaining predict  $\Delta MR$  and use the head core temperature to represent  $T_c$ . The original parameter values (P1, P2, etc.) were converted, if necessary, to conform to metabolic rates in W and are listed in Tables 2 and 3.

The STRONG model (7) was originally applied to groups of low and high BF with separate parameter values (see Table 3). It represents the simplest model possible with  $T_{sk}$  and  $T_c$ , and is closely related to the TIMBAL model (6). The

original form of the TIMBAL model was re-arranged to eliminate unnecessary parameters (with respect to regression analysis). It is the only model listed to use the subject's  $A_D$  and the rate of change of mean skin temperature ( $\dot{T}_{sk}$ ). The HAYWARD model (9) is the only one listed to use the subject's WT. The inclusion of  $A_D$  and WT into these models will be examined for their affect on the predictive capability of the models.

The WISSLER model (11) expanded upon the HAYWARD model by including the subject's rate of change of core temperature ( $\dot{T}_c$ ). The form shown in Table 1 is a simplified version of the original model and is applicable when  $\Delta MR$  changes slowly, as was the case with the present data. The original model also included a term with  $\dot{T}_{sk}$ ; however, this term decays exponentially with a half-time of 30 s when  $\dot{T}_{sk} > -1.5^\circ C/min$ , which the data we used did not include, so this term was intentionally left out.

The NADEL model (8) is similar to the HAYWARD model except for an additional term involving only  $T_{sk}$ . This term was added (8) to describe the observed metabolic response without the necessity of changing the hypothalamic set-point temperature or P3 in the model. The STOLWIJK model (10) emphasized the contribution of this additional term by introducing an exponent of 2. Both models consider the parameters P2 and P3 as physiological set-points, to be identified with temperatures of "standard" man under a condition of thermal neutrality.

The PRESENT model is based on the STOLWIJK model with the modification that the set-point values ( $T_{sk, set}$  and  $T_{c, set}$ ) were assigned the average of the measured pre-immersion values and remained fixed. For example,  $T_{sk, set}$  of a particular group was the average of all pre-immersion values of  $T_{sk}$  for that group. This procedure allowed the use of "personalized" set-points which did not necessarily match the values of "standard" man. For data used in this study,

$T_{sk, set}$  ranged from 32.79 to 33.56°C and  $T_{c, set}$  ranged from 37.14 to 37.39°C; these ranges fell outside the values 37° and 36.96°C, respectively, used for "standard" man (10).

Since the data we used provided rectal temperature as the only measured core temperature, this temperature was applied in place of the head core temperature of the WISSLER, NADEL, STOLWIJK and PRESENT models. In these circumstances, the functional form of these models and not necessarily their original parameter values were tested. In anticipation that an inverse relationship between BF and the prediction of the metabolic response for a given lowered  $T_{sk}$  and lowered  $T_c$  exists, all models were modified "a priori" by dividing by  $(\%BF)^x$  where  $x$  is an estimated parameter. These modified versions are designated by the affix BF-modified.

Analysis. A regression analysis was performed on all models using the BMDP statistical package (16). Significant ( $\alpha = 0.05$ ) improvement in the prediction of a particular model when parameter values changed or when a parameter was added to the model was tested using Fisher's F-test as outlined by Mekjavić and Morrison (17). Significance between predictions of the models listed in Table 1 could not be tested since the models were fundamentally different from one another. However, in these cases, predictions were compared by the models' sum of squared residuals (SSR) given by

$$SSR = \sum_{n=1}^N w_n e_n^2 \quad (8)$$

where  $N$  is the number of cases (grouped observations),  $w_n$  is the case weight (equal to 1 for groups of 4 subjects and 1.25 for groups of 5 subjects) and  $e_n$  is the residual or difference between the observed and predicted value. In essence, the

SSR is a measure of how well the model prediction fits the data. Models with the lowest SSR values were deemed better predictors than those of higher SSR values, although no significance to these comparisons could be given.

The residuals of the model that predicted best overall were analyzed graphically as outlined by Draper and Smith (18). For convenience, the "unit normal deviate" form ( $e_n/s$ ) of the residuals was plotted where  $s$  is the square root of the residual mean square given by

$$s = \sqrt{\text{SSR}/df} \quad (9)$$

and df is the number of degrees of freedom (equal to  $N - p$  where  $p$  is the number of parameters estimated).

To establish whether a BF dependency in the prediction of MR and ΔMR exists, each model in their unmodified form was applied to the LBF and HBF groups separately, similar to the way the STRONG model was originally applied. Because the variability of BF within these subgroups is small, any dependency of the metabolic response upon BF, if it exists, is effectively removed. Therefore, the resultant SSR should then approximate the lowest possible value without introducing any new parameters. To compare these SSR values with those obtained by treating the combined data (LBF+HBF) when using the same unmodified model, the sum of both groups, i.e.,  $\text{SSR}(\text{LBF}) + \text{SSR}(\text{HBF})$ , was used so that all the residuals were accounted for. If little difference between these two values was found, then it would be concluded that BF is not a critical variable in the prediction of metabolic response for a given lowered  $T_{sk}$  and lowered  $T_c$ . However, if a large difference was found, then BF would be considered a critical variable and its role could then be quantified through modifications of the models such as described by the BF-modified versions. An appropriate version is one in which using the combined data results in a value of  $\text{SSR}(\text{LBF+HBF})$  close to the sum of  $\text{SSR}(\text{LBF}) + \text{SSR}(\text{HBF})$  using the separated data as described above.

## RESULTS

Comparison Between Original and Regressed Estimates for Combined Data. Table 2 lists the model's (a) original (where applicable) and (b) regressed parameter estimates and their corresponding SSR when applied to the combined data of the LBF and HBF groups. The predictions improved significantly for all models (where original parameter estimates were given) using the regressed parameter estimates. The following comments pertain to the regressed versions. The subscripted alphabet in parenthesis affixed to SSR refers to the corresponding set of parameter estimates listed in Tables 2 and 3. Among the regressed versions, the WISSLER and PRESENT models predicted best and the HAYWARD model predicted worse (see  $SSR_{(b)}$  values plotted in Fig.1).

To check the role of  $A_D$  in the prediction of MR in the TIMBAL model, this variable was removed and the subsequent regression led to a SSR value of 127,773. This value is smaller than the value 133,416 obtained by leaving  $A_D$  in the model. Thus, using the present data, the inclusion of  $A_D$  worsened the predictive capability of the TIMBAL model. A similar result was found when the variable WT was removed from the HAYWARD model. In this case, the resultant SSR was 123,146, considerably smaller than the value 158,953 obtained by leaving WT in the model.

As a further check, the variables  $A_D$  and WT were introduced independently as factors to the PRESENT model equation to examine whether the inclusion of these variables could improve this model's predictive capability. The subsequent regression by including  $A_D$  led to a SSR value of 84,694 and that obtained by including WT was 95,112. Both values exceeded the SSR value of 83,150 obtained when neither of these variables was used, thus no improvement was achieved by their inclusion into the model.

A very high correlation ( $r > 0.99$ ) was found between P1 and P4 of the TIMBAL model, suggesting that  $T_{sk}$  was an unnecessary variable to include for the present data. This is further substantiated by noting that the SSR value obtained by removing  $A_D$  from the model was not significantly different from the value of 128,615 obtained using the STRONG model which is identical in form except for the inclusion of the  $T_{sk}$  term.

A linear dependency was found between the parameters of the NADEL model. This was not unexpected since the model equation (see Eq.5) could have been rewritten without the second term such that  $P3(\text{new}) = P3 + P4/P1$ . This simplified form was not used by Nadel et al. (8) for reasons stated earlier but was proposed by Montgomery (19), and when applied to the regression analysis, it was found to yield the same SSR. A marginal improvement over the NADEL model was obtained using the STOLWLUK model. Because of the exponent of 2 raised on the second term of this model, no linear dependency of the parameters was found.

Comparison Between Using Combined Data and Separated Data. Table 3 lists the model's (a) original (applicable only in the case of the STRONG model) and (d) regressed parameter estimates and their corresponding SSR when applied to the data of the LBF and HBF groups separately. The prediction of the STRONG model was improved significantly using the regressed parameter estimates over the original parameter values. The SSR values of the HBF group were consistently lower than those for the LBF group for all models indicating a greater variability in the metabolic response for the LBF groups.

To compare these results with those obtained by treating the combined data, the sums of  $\text{SSR}_{(d)}(\text{LDF}) + \text{SSR}_{(d)}(\text{HBF})$  are plotted alongside  $\text{SSR}_{(b)}(\text{LBF+HBF})$  in Fig.1. In all cases, the former sum is substantially smaller. This result confirms that the predicted metabolic response to a lowered  $T_{sk}$  and lowered  $T_c$  has a BF dependency.

Functional Role of BF. Dividing each model by  $(\%BF)^x$  (i.e., BF-modified version) significantly improved the prediction over the unmodified versions using the combined data in all cases. Furthermore, the  $SSR(LBF+HBF)$  values obtained this way were close to the sum of  $SSR_{(d)}(LBF) + SSR_{(d)}(HBF)$  indicating that an inverse relationship of BF to metabolic response was appropriate to consider. The regressed estimates of  $x$  varied among the models, but for practical purposes, rounded values are presented. These are 0.5 for the models that predict MR and 1.0 for the models that predict  $\Delta MR$ . The regressed parameter estimates of the BF-modified models using these exponents and the corresponding  $SSR(LBF+HBF)$  are listed in Table 2. These SSR values are also shown in Fig. 1. Rounding-off the exponent  $x$  was possible because of an asymptotic standard deviation of about 10% in the estimate of  $x$  and because of a high correlation between  $x$  and some of the other model parameters.

Residual Analysis of the PRESENT BF-Modified Model. A residual analysis of the PRESENT BF-modified model was conducted since this model provided the lowest SSR. Figure 2 shows the normalized residuals plotted against the a) predicted values of AMR, b) time, c)  $T_{sk}$  and d)  $T_c$ . Since 95% of the normalized residuals fell within the limits (-1.96, 1.96), this distribution was assumed normal (18). In addition, no apparent trend is evident in the plot of residuals against the predicted values of  $\Delta MR$ ; therefore, the residuals were also considered randomly scattered.

When the normalized residuals of the nude and clothed groups plotted against time in Fig. 2(b) are considered separately (recall that the nude immersions lasted 1 h and the clothing immersions lasted 3 h), the residuals are randomly scattered; that is, no time dependency is apparent. The plot of residuals against  $T_{sk}$  shown in Fig. 2(c) revealed a slight tendency from overprediction at low  $T_{sk}$  to underprediction at high  $T_{sk}$ . This tendency can, if desired, be reduced without

compromising the randomness of the other plots by using a smaller exponent in place of 2 for the  $T_c$  term in the BF-modified version of Eq.7. The final plot, Fig.2(d), of residuals against  $T_c$  indicated random scatter. These results suggest that anomalies are few and small; hence further modification of the model offers little benefit and was not pursued.

## DISCUSSION

The results suggest that an individual's metabolic response to a lowered  $T_{sk}$  and lowered  $T_c$  is attenuated by his %BF. The physiological mechanism underlying this attenuation is beyond the scope of the present study. However, this attenuation is consistent with the fact that increased BF promotes thermal insulation (1-3) and, therefore, at a given lowered  $T_{sk}$  and lowered  $T_c$ , less heat production is required to maintain deep body temperature.

The wide disparity between the original parameter values and the regressed parameter estimates for the unmodified version of all models may be explained, in part, by differences in the experimental conditions from which data were obtained to derive these values. Strong et al. (7) used data of whole body immersion in water temperatures between 20 and 36°C. Timbal et al. (6) and Hayward et al. (9) used data of immersions to neck-level in water temperatures of 15 and 26°C, and 10°C, respectively, and Nadel et al. (8) used data from exposure to cold air. Differences in data may also explain why the regressed parameter estimates proposed by Mekjavić and Morrison (17) for the HAYWARD model differed widely from those we found. The data used by Mekjavić and Morrison (17) involved immersions of lean individuals in 10°C water. These authors found the residuals, when plotted against  $T_{sk}$ , displayed a shape that was characteristic of the average discharge frequency of cold receptors in the skin. This bell-shape with a maximum at about 25°C is not apparent in Fig.2(c) where  $T_{sk}$  ranges from 21 to 31°C. To confirm

that this result was model-independent, a residual analysis on the predictions using the original HAYWARD model with the regressed parameter estimates (set (b) in Table 2) did not reveal any bell-shape distribution when plotted against  $T_{sk}$ .

The addition of the  $T_{sk}$  term in the TIMBAL model did not improve this model's predictive capability when compared to the results of the STRONG model which is similar but excludes the  $T_{sk}$  term. We are uncertain about the value of  $T_c$  used in the WISSLER model despite its good predictive performance because of the unusual regressed parameter estimates. It is interesting that the TIMBAL and HAYWARD models predicted better without the inclusion of the  $A_D$  and WT variables, respectively, suggesting that these variables are superfluous in the prediction of MR using the present data. This finding was further supported when  $A_D$  and WT were introduced independently in the PRESENT model and its predictive capability worsened in both cases.

The functional form of the HAYWARD model is the same as the NADEL model considering that the second term of the NADEL model can be left out without affecting its predictive capability, yet there is a large difference between their SSR values. This difference is attributed partly to the inclusion of WT in the HAYWARD model recalling that the SSR decreased from 158,953 to 123,146 when WT was removed from the model. The remaining difference is largely attributed to the prediction of MR on the one hand and ΔMR on the other. This is because the variability in the initial value of MR is present when predicting MR, as in the case of the HAYWARD model, but this variability is removed when predicting ΔMR (recall that ΔMR is the increase in metabolic rate above the measured pre-immersion value). Greater accuracy can therefore be expected from models that avoid this initial variability. This is further supported by the relatively poor predictions of the other two models, STRONG and TIMBAL that also predict MR.

This difference in how the metabolic response is predicted also helps to explain why the square root inverse relationship works best for models predicting MR and why the unit inverse relationship of BF works for models predicting  $\Delta$ MR. For the same increase in metabolic rate, the relative change in  $\Delta$ MR is larger than for MR. It follows, therefore, that relative differences in  $\Delta$ MR between the LBF and HBF groups are also larger than relative differences in MR between these subgroups. These differences can be adequately balanced by dividing the original models that predict  $\Delta$ MR by %BF and those that predict MR by  $\sqrt{\%BF}$ .

### CONCLUSIONS

Regressed parameter estimates for all unmodified models improved the prediction of metabolic responses significantly over the original parameter values. This suggests that when these empirical models are used, the values of the parameters should correspond to exposure conditions from which they were derived. In addition, since the esophageal temperature represents more closely the head core temperature than does the rectal temperature, the predictive capabilities of the WISSLER, NADEL, STOLWIJK and PRESENT models may improve with the use of the esophageal temperature, if available.

Applying each model to the LBF and HBF groups separately demonstrated that BF is a critical variable to consider when predicting the metabolic response for a given lowered  $T_{sk}$  and lowered  $T_c$ . Applying an inverse relationship of %BF to the metabolic response (i.e., the BF-modified versions) significantly improved all the models' predictive capability. The PRESENT model predicted best overall, both in the unmodified form when applied to the LBF and HBF groups separately and in the BF-modified form when both groups were combined. The best alternative if specific thresholds or "personal" set-points were unavailable was the STOLWIJK model using the regressed parameter estimates given in Tables 2 and 3.

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**FIGURE CAPTIONS**

1. Sum of squared residuals for the combined data using the regressed parameter estimates ( $SSR_{(b)}(LBF+HBF)$ ), for the separated data using the regressed parameter estimates ( $SSR_{(d)}(HBF)$ ), and for the combined data using the BF-modified regressed parameter estimates ( $SSR_{(c)}(LBF+HBF)$ ).
2. Normalized (unit normal deviate form) residuals of the PRESENT BF-modified model plotted against (a) predicted increase in metabolic rate, (b) time, (c) mean weighted skin temperature and (d) core temperature. The closed circles represent the nude condition and the open circles represent the clothed condition.

FIG. 1

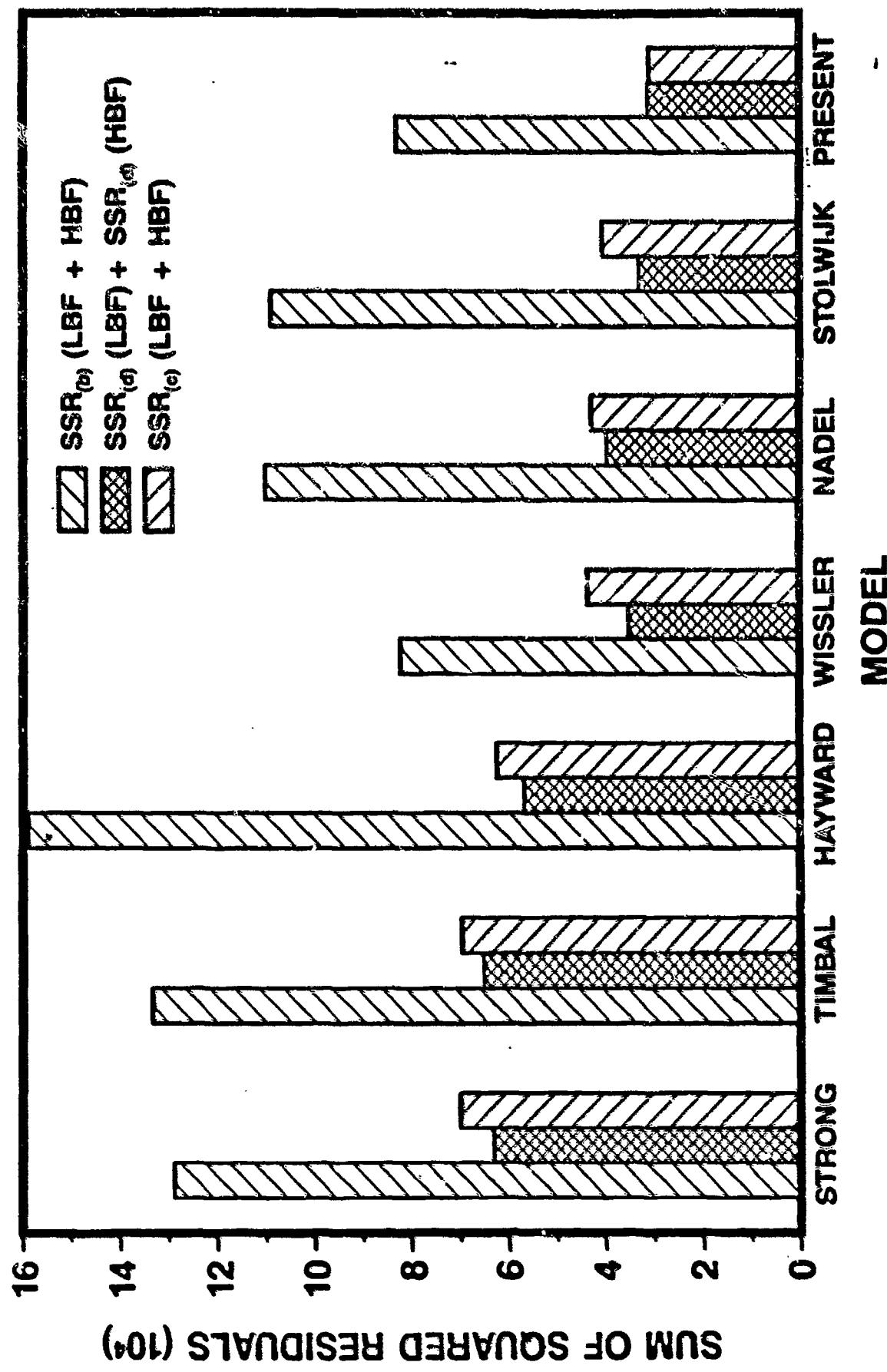


Fig. 2

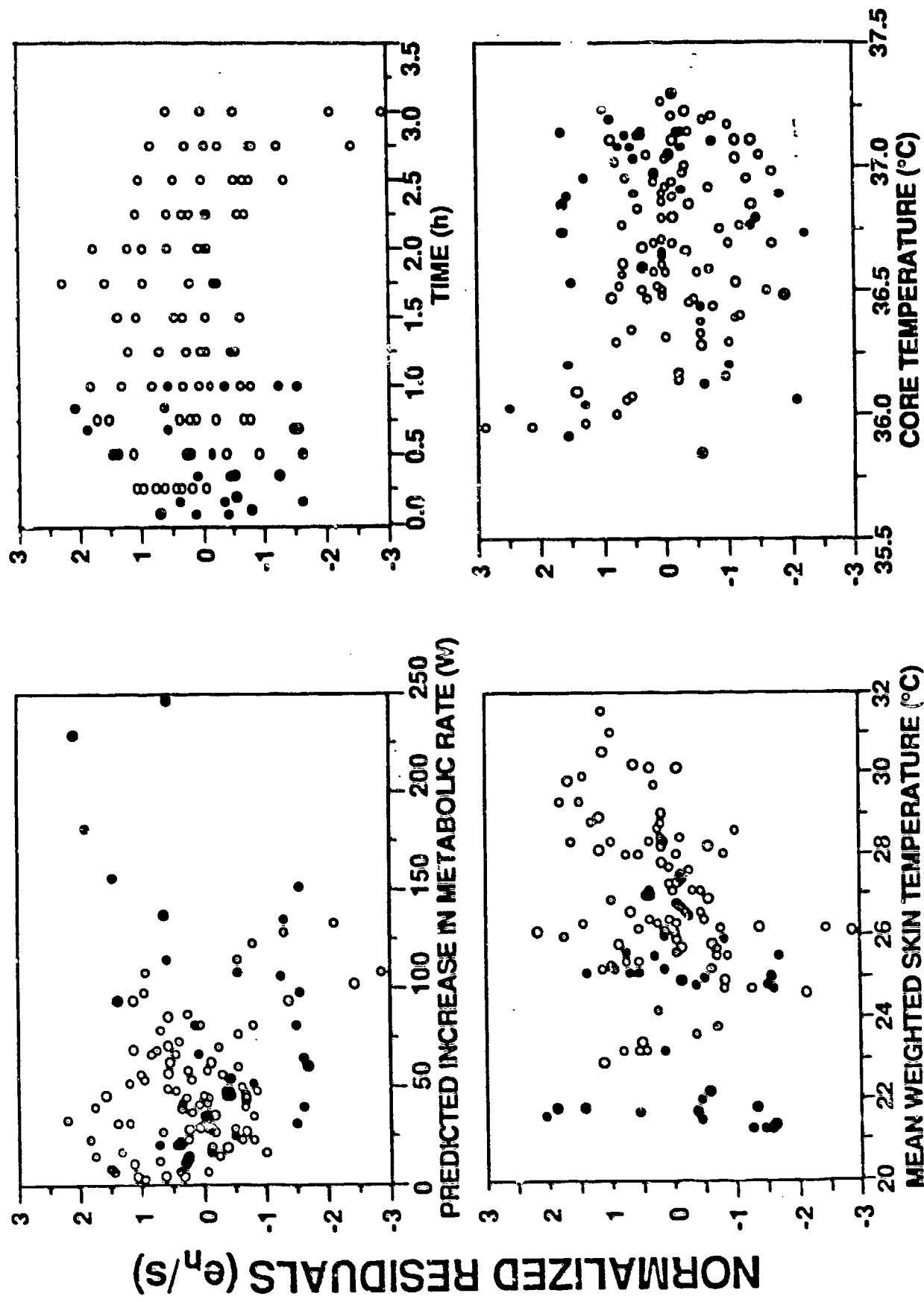


Table 1: Empirical Models for Predicting Metabolic Response for Lowered  $T_{sk}$  and Lowered  $T_c$

STRONG	$MR = P1 - P2 * T_{sk} - P3 * T_c$	(1)
TIMBALI	$MR = A_D * (P1 - P2 * T_{sk} - P3 * T_c - P4 * f_{sk})$	(2)
HAYWARD	$MR = WT * P1 * (T_{sk} - P2) * (T_c - P3)$	(3)
WISSLER*	$\Delta MR = P1 * (T_{sk} - P2) * (T_c - P3) - P4 - P5 * (f_c + 0.01)$	(4)
NADEL	$\Delta MR = P1 * (P2 - T_{sk}) * (P3 - T_c) + P4 * (P2 - T_{sk})$	(5)
STOLWIK	$\Delta MR = P1 * (P2 - T_{sk}) * (P3 - T_c) + P4 * (P2 - T_{sk})^2$	(6)
PRESENT	$\Delta MR = P1 * (T_{sk, set} - f_{sk}) * (T_{c, set} - T_c) + P2 * (T_{sk, set} - f_{sk})^2$	(7)

\* if  $T_c > 36.8^\circ C$ , then set  $T_c = 36.8^\circ C$  and if  $f_c > -0.01^\circ C/min$ , then set  $P5 * (f_c + 0.01) = 0$

**Table 2: Original (a), Regressed (b) and SF-Modified (c) Parameter Estimates and SSR for Combined Data**

<u>Model</u>		<u>P1</u>	<u>P2</u>	<u>P3</u>	<u>P4</u>	<u>P5</u>	<u>SSR</u>
<b>STRONG</b>	(a)	2414	6.1	65.6	-	-	128,916
	(c)*	6169	37.7	126.4	-	-	78,647
<b>TINBAL</b>	(a)	1186	5.0	23.6	57.6	-	373,214
	(b)	1229	4.5	26.7	16.9	-	183,416
	(c)*	8846	21.6	59.2	-26.8	-	69,736
<b>HAYWARD</b>	(a)	6.6214	42.4	41.4	-	-	214,627
	(b)	6.6447	42.7	39.2	-	-	158,963
	(c)*	6.1327	41.6	48.6	-	-	62,453
<b>WEISSLER</b>	(a)	2.71	41.6	41.6	97.7	1847	292,661
	(b)	9.21	44.4	37.6	-14.1	8932	82,461
	(c)	162.1	46.7	37.3	-29.6	14184	43,862
<b>NADEL</b>	(a)	41.9	92.2	86.6	8.1	-	468,687
	(b)	9.8	22.6	30.5	16.4	-	118,167
	(c)	93.5	21.5	36.8	11.9	-	42,976
<b>STOLWJK</b>	(a)	13.9	32.97	36.96	8.4	-	152,936
	(b)	7.1	34.28	37.34	8.24	-	106,973
	(c)	71.9	32.41	37.48	6.1	-	46,719
<b>PNESENT</b>	(b)	8.2	6.84	-	-	-	83,169
	(c)	75.8	6.4	-	-	-	36,949

\* models modified by - KBF, others modified by - XBF

Table 3: Original (a) and Regressed (d) Parameter Estimates  
and SSR for Separated Data

<u>Model</u>	<u>Group</u>	<u>p1</u>	<u>p2</u>	<u>p4</u>	<u>p6</u>	<u>SSR</u>
STRONG	LBF	{a}	1161	19.8	11.6	267,638
	HBF	{d}	1913	16.2	35.6	49,769
			629	15.1	6.4	661,864
			1426	5.4	31.2	13,236
TIMBAL	LBF	{d}	312	9.1	15.7	51,847
	HBF	{d}	751	8.3	17.1	-10.3
					6.3	13,317
HAYWARD	LBF	{d}	0.6569	37.1	41.8	43,672
	HBF	{d}	0.0177	47.5	41.0	13,248
WISSLER	LBF	{d}	16.8	29.1	37.3	2228
	HBF	{d}	0.7	36.7	37.2	-1671
NADEL	LBF	{d}	19.6	39.3	36.2	29,741
	HBF	{d}	8.8	34.7	36.0	10,661
					7.2	
STOLWIJK	LBF	{d}	8.7	32.74	37.22	23,167
	HBF	{d}	8.6	35.45	37.74	9,994
PRESENT	LBF	{d}	6.6	37.0	-	21,749
	HBF	{d}	4.6	37.0	-	9,465